Influence of Perindopril on Left Ventricular Global Performance During the Early Phase of Inferior Acute Myocardial Infarction: Assessment by Tei Index

Nearchos S. Nearchou, M.D., Alexandros K. Tsakiris, M.D., Maria D. Lolaka, M.D., Ilias Zarcos, M.D., Dimitrios P. Skoufas, M.D., and Panagiotis D. Skoufas, M.D.

First Cardiology Department, Hellenic Red Cross Hospital, Athens, Greece

The beneficial effect of angiotensin-converting enzyme inhibitors (ACE inhibitors) on left ventricular (LV) function in patients with acute myocardial infarction (AMI) is widely known. However, controversy exists about their efficacy on patients with small infarcts and preserved LV systolic function. The aim of the present study was to detect the influence of the ACE-I perindopril on the global LV performance in patients with pure inferior AMI (AMI-I) using a Doppler-derived index (DI) that combines systolic and diastolic time intervals (Tei index). Our study included 40 patients with first AMI-I, mean age 60 years ±9.06 years (SD) and 24 age- and gender-matched normal patients who constituted the control group (COG). Patients were randomized into two groups to receive the conventional treatment of AMI-I (GCT) or the above therapy plus P (GP). Complete Doppler echocardiography (systolic and diastolic parameters), DI, and systolic blood pressure (SBP) were measured on the 8.07 ± 1.16 (SD) post-infarct day. The same examination was performed to COG. The DI was significantly lower in healthy patients (0.45 ± 0.23) compared with the value in patients of either GP (0.56 ± 0.03; P = 0.023) or GCT (0.78 ± 0.05; P = 0.000). Moreover DI was higher in patients of GCT compared with that of GP (P = 0.000). In addition, perindopril administration decreased isovolumic relaxation time (IRT; 120.00 ± 4.23 vs. 139.00 ± 6.74; P = 0.006) and increased significantly ejection time (ET; 274.25 ± 7.35 vs. 253.50 ± 7.68; P = 0.042). SBP in patients of GP was similar to that of GCT (120.5 ± 2.85 mmHg vs.112.5 ± 3.49 mmHg; P = NS). Conclusions: Global LV function (DI) is impaired in patients with AMI-I. Administration of perindopril has a favorable impact on LV performance in patients with AMI-I, achieved through improvement of the diastolic function (IRT), which indirectly improves LV systolic function (ET, DI). This beneficial influence of perindopril is the result of the direct tissue effect of the drug and not its hemodynamic action. (ECHOCARDIOGRAPHY, Volume 20, May 2003)

inferior acute myocardial infarction, *perindopril*, *Doppler-index of overall myocardial performance*, *Tei index*

In the past two decades, angiotensin-converting enzyme inhibitors (ACE inhibitors) were established as an important addition to the list of treatments of myocardial infarction (MI). Extensive multicenter studies such as GISSI-3, SAVE, AIRE, ISIS-4, SOLVD, and many others[1–23] have reported favorable influence of ACE inhibitors on patients with MI. Evidence from these studies indicates that during the early phase of MI, ACE inhibitors limit infarct expansion[7] and left ventricular (LV) enlargement,[9–11,12,16,17,20] and during the post-infarct period they reduce the mortality[1,4,5,18,19,21,22] and the rate of development of heart failure.[7,9,18,19,22] These beneficial results are more obvious in patients with extensive acute myocardial infarction (AMI)[7,13,14] accompanied by LV dysfunction[1,5,19,21] and clinical manifestations of LV failure.[1,7,13,14,21,22]

Nevertheless, there has not been a complete agreement among researchers concerning the existence of a favorable influence of ACE inhibitors on patients with limited extension AMI and preserved LV function.[13,14] This study has

Address for correspondence and reprint requests: Nearchos S. Nearchou, M.D., 50 Vlachaki St., 115 25, Athens, Greece. Fax: +30 6716989; E-mail: a040144@otenet.gr

been focused on the detection of the affect of ACE inhibitor perindopril, in patients after their first uncomplicated acute inferior wall myocardial infarction (AMI-I). At the time of writing, PREAMI is the first trial that evaluates efficacy of long-term ACE inhibition with perindopril in patients 65 years old, with AMI and preserved LV systolic function.[24]

Although AMI is a process resulting in global dysfunction of LV, in the previously mentioned studies only systolic parameters[7,9,11,19–22] and especially the ejection fraction have been used for assessment of LV function after AMI. The ejection fraction is a simple and reliable index only of systolic cardiac performance. Moreover, there is not yet an available ideal method for easy and precise determination of LV diastolic dysfunction. Although the standard methods of Doppler echocardiography (assessment of isovolumetric relaxation time [IRT], transmitral flow,[25,26] and pulmonary venous flow[27–29]) seem to predominate over cardiac catheterization and radionuclide ventriculography for the evaluation of LV diastolic dysfunction, they are subject to restrictive dependence on heart rate,[30] breathing,[31] age,[32] blood pressure, preload,[33] and afterload. Furthermore, it is hypothesized that a measure of combined systolic and diastolic myocardial performance is a better predictor of overall LV function than systolic or diastolic parameters separately.

Consequently, the existence of a clinically applicable method for assessment of global myocardial performance in patients with AMI would be very useful. Such a method is the systolic-diastolic Doppler Index (DI) or Tei index, which has been studied in several cardiac diseases[34–44] and has been proved to have both predictive value[34,37,44] in patients with LV dysfunction and good correlation with global LV performance.[34,35] Moreover, there is evidence that this index is more sensitive than mitral E, A waves and deceleration time of E wave for the assessment of LV diastolic function and has a better correlation with—dp/dt and tau.[45] In our previous studies, we used this method in patients with AMI, and we were the first to demonstrate the abnormal deviation and predictive value of the index in this population.[46,47] Recently Poulsen et al.[48] confirmed this deviation of DI in the early postinfarct phase and revealed its positive correlation with the risk of development of congestive heart failure.

On these grounds, our study aims to evaluate the influence of perindopril on global LV function in patients with pure AMI-I focusing on the utility of DI. This index is easily obtained, noninvasive, and independent of blood pressure,[34,35,49] heart rate,[34,35,49,50] age,[50] sex,[50] and ventricular geometry.

Methods

This study included 40 patients (28 males), mean age 60 ± 9.06 (SD) years with first AMI-I. This was diagnosed on the basis of the following criteria: characteristic chest pain for MI, electrocardiogram changes favoring limited inferior infarction, and diagnostic serial changes in cardiac enzymes.

The thrree groups were as follows: group GCT comprised 20 patients receiving the conventional therapy for AMI-I; group GP comprised 20 patients receiving, in addition to the conventional treatment, perindopril, in a dose of 2 to 4 mg once per day, beginning on the third postinfarct day; and group COG comprised 24 age- and gender-matched normal patients as control group. Healthy individuals had no history or symptoms suggestive of cardiovascular disease and normal findings on physical examination, electrocardiography, and echocardiography. Titration of perindopril in GP patients was in proportion to their basic systolic blood pressure (SBP) in order to achieve better tolerance and lower hypotensive complications. All patients underwent M-mode, two-dimensional, and Doppler echocardiography on the 8.07 ± 1.16 (SD) postinfarct day. The same examination was also performed on all healthy patients. DI, conventional echocardiography, and Doppler derived systolic and diastolic parameters were calculated for all patient in fasting condition and at midday.

In our study therapy with perindopril was instituted on the third postinfarct day. Available data indicate that previous selective trials[1,21,22] initiated treatment with ACE inhibitors between 3 and 16 days after AMI and maintained it for 1 to 4 years, whereas unselective trials[4,23] initiated this therapy within 24 to 36 hours and maintained it for 4 to 6 weeks. A substantial portion of the lives saved occurred within the first several days after MI. Believing that a more acceptable risk/benefit ratio might have been achieved we delayed the initiation of treatment with perindopril until the third postinfarct day, when the frequency of significant hypotension would be considerably reduced.[5,21] Moreover, we decided to calculate the DI on the eighth postinfarct day when diastolic

dysfunction was assumed to have taken place. It is well known that the initial inciting event after coronary artery occlusion is an abrupt loss of contractile tissue and LV systolic dysfunction appears to predominate in the early phase of MI. LV diastolic properties modification appears later because of compensatory hypertrophy and ventricular remodeling process.[51–53] This provided to us a window of opportunity to assess the impact of perindopril on LV remodeling at the time that diastolic dysfunction is apparent.

Conventional treatment for AMI-I was considered the administration of -blockers, nitrates, heparin, and aspirin. Both groups of patients were treated with the same pharmaceutical regimen of each drug category. Intravenous administration of nitrates and heparin were discontinued on the third postinfarct day.

Thrombolysis was administered in 23 (57%) of 40 patients with AMI-I. Patients in both groups were comparable regarding history of hypertension and thrombolytic therapy. Individuals with relative or absolute contraindications to ACE inhibitors, persistent (>1 hour) severe hypotension (SBP < 100 mmHg), history of old MI, or percutaneous transluminal coronary angioplasty or coronary artery bypass grafting, coexistent right ventricular infarction, atrial fibrillation, left bundle branch block, sustained angina pectoris, postinfarct mechanical complication, or any valvulopathy more severe than mild, were excluded from the study.

Complete two-dimensional, M-mode, and Doppler echocardiograms were performed using a commercially available ultrasound instrument (ATL-UM 9; Advanced Technology Laboratories, Bothell, MA, USA) with a phased array transducer of 2.5 MHz. We used the parasternal long-axis view at the mid-LV level to measure M-mode LV end-systolic and end-diastolic diameters. From the same view at the aortic level, atrial diameter was assessed. Ejection fraction was calculated using Bullet's formula. The mitral inflow velocity pattern was recorded from the apical four-chamber view with the pulsed-wave Doppler sample volume positioned at the tips of the mitral leaflets in the center of the flow stream during diastole.[54] The following parameters were measured at a recording speed of 100 mm/s: transmitral peak rapid filling velocity (E wave); peak atrial filling velocity (A wave); E-wave deceleration time-peak to zero-(DT); and E/A ratio. The LV isovolumetric relaxation time (IRT) was obtained from the apical five-chamber view by simultaneous recording of the aortic outflow and mitral inflow. This interval was measured from the end of aortic outflow to the beginning of the mitral inflow at a recording speed of 100 mm/sec.[26,54–56] These parameters for assessment of LV diastolic function are accepted in the bibliography as having a good correlation with radionuclide and angiographic techniques.[25,57] Additionally, from the apical five-chamber view, with the pulsed-wave Doppler sample volume positioned just below the aortic valve, we recorded the LV outflow pattern. The following systolic parameters were measured: isovolumetric contraction time (ICT), which is the interval from the closure of the mitral valve to the opening of the aortic valve; and LV ejection time (ET), which is the duration of LV outflow velocity profile.

Finally, we obtained the Tei index, combining Doppler systolic and diastolic time intervals measured from mitral inflow and LV outflow velocity time patterns as demonstrated in Figure 1. The interval a from the cessation to the onset of mitral inflow was equal to the sum of ICT, ET, and IRT. The interval b was the duration of LV outflow velocity profile. Thus, the sum of ICT and IRT is obtained by subtracting

Doppler-Index= (a-b) / b = (ICT+IRT) / ET

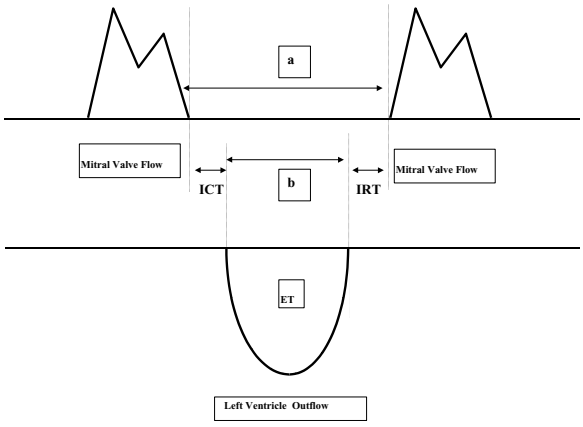

Figure 1. *Scheme for measurements of Doppler time intervals. The index is defined as (a–b)/b, where a is the interval between cessation and onset of the mitral inflow and b is the ejection time (ET) of left ventricular outflow. Isovolumetric relaxation time (IRT) is the interval between the aortic valve closure and the onset of mitral valve flow. Isovolumetric contraction time (ICT) is the interval between the cessation of mitral inflow and the onset of aortic valve flow.*

b from a. The index of combined LV systolic and diastolic function (the sum of ICT and IRT divided by ET) was calculated as (a–b)/b.

All Doppler measurements were calculated at the end of expiration from an average of five consecutive cardiac cycles.

Statistics

The Student's *t*-test for independent samples method was used for comparison of quantitative observations and[2] to compare qualitative characteristics. A P value of <0.05 was considered statistically significant.

Results

Clinical Characteristics and General Echocardiographic Findings of Groups Studied

Table I presents the clinical characteristics and general echocardiographic findings of groups studied. The three study groups were comparable regarding age, gender, heart rate, diastolic blood pressure, and LV end-diastolic diameter. SBP was higher in healthy patients compared with the two patient groups. Moreover, the SBP of GP patients was insignificantly higher than that of GCT patients (P = 0.072). As was expected, the ejection fraction was significantly higher in healthy patients compared with the value in the two patient groups, but it was similar in the two patients groups and within normal limits.

Values of Doppler Parameters and Tei Index in Groups Under Investigation

The ICT was significantly shortened in normal patients compared with GCT patients, but there was no difference between healthy patients and GP patients (P = 0.555). Moreover, ICT was marginally shorter in GP patients compared with GCT patients (P = 0.099; Fig. 2, Table II). The IRT was significantly shorter in healthy patients compared with the value in the two patient groups. Furthermore, this time interval was prolonged in GCT patients compared with that in GP patients (Fig. 2, Table II). ET was significantly prolonged in healthy patients compared with the patient groups. In addition, GP patients exhibited longer ET than GCT patients (Fig. 2, Table II). Concerning ratio E/A, it was higher in normal patients than in the two patient groups, but there was no significant difference between the two groups of patients (P = 0.769; Table II). The DT was insignificantly different between normal patients and GP patients (P = 0.074). Additionally, this interval was shorter in normal patients compared to GCT patients and similar between the two groups of patients (P = 0.738; Table II).

The index was significantly lower in age- and gender-matched healthy patients (0.45 ± 0.23) compared with that in GP patients (0.56 ± 0.03; P = 0.023) and also with that in GCT patients (0.78 ± 0.05; P = 0.000), whereas it was significantly higher in GCT patients compared with

Table I

Comparison of the Basic Clinical Characteristics and the General Echocardiographic Findings of Subjects in the Three Groups of Study: 24 Healthy Subjects as Control Group (COG), 20 Patients with AMI-Inferior (AMI-I) Receiving Conventional Treatment Plus Perindopril (GP) and 20 Patients with AMI-I Receiving Only Conventional Treatment (GCT).

	COG (MV ± SE) n = 24	GP (MV ± SE) n = 20	P	COG (MV ± SE) n = 24	GCT (MV ± SE) n = 20	P	GP (MV ± SE) n = 20	GCT (MV ± SE) n = 20	P
Age (years)	59.62 ± 1.51	58.85 ± 2.21	NS	59.62 ± 1.51	61.15 ± 1.86	NS	58.85 ± 2.21	61.15 ± 1.86	NS
Female/Male	10/14	4/16	NS	10/14	8/12	NS	4/16	8/12	NS
Heart rate (b/m)	67.2 ± 3.02	70.6 ± 2.55	NS	67.2 ± 3.02	71.05 ± 3.99	NS	70.6 ± 2.55	71.05 ± 3.99	NS
Systolic BP (mmHg)	129.58 ± 2.67	120.5 ± 2.85	0.034	129.58 ± 2.67	112.5 ± 3.49	0.000	120.5 ± 2.85	112.5 ± 3.49	0.072
Diastolic BP (mmHg)	78.75 ± 1.25	74.75 ± 1.94	NS	78.78 ± 1.25	75.50 ± 2.46	NS	74.75 ± 1.94	75.5 ± 2.46	NS
LVd (cm)	4.97 ± 0.12	4.94 ± 0.11	NS	4.97 ± 0.12	4.91 ± 0.13	NS	4.94 ± 0.11	4.91 ± 0.13	NS
L Ad (cm)	3.63 ± 0.08	4.00 ± 0.07	0.003	3.63 ± 0.08	4.07 ± 0.09	0.005	4.00 ± 0.07	4.07 ± 0.09	NS
Ejection fraction (%)	73.7 ± 1.39	59.8 ± 2.16	0.000	73.7 ± 1.39	56.2 ± 3.21	0.000	59.8 ± 2.16	56.2 ± 3.21	NS

AMI = Acute Myocardial Infarction, BP = blood pressure, LVd = Left ventricular end-diastolic diameter, LAd = left atrial dimension, p = statistical significance, NS = Statistically not significant. $p < 0.05$ statistically significant. MV ± SE = mean value ± standard error.

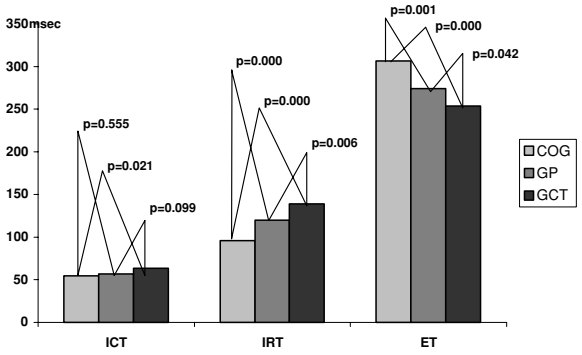

Figure 2. *Comparison of Doppler time intervals between normal patients (COG), patients who received the conventional treatment (GCT) of acute inferior myocardial infarction (AMI-I), and patients who received perindopril in addition to conventional treatment (GP). Left ventricular (LV) isovolumetric relaxation time (IRT) was significantly prolonged in patients with AMI-I compared with that in normal patients, whereas the administration of perindopril shortened this time interval. LV isovolumetric contraction time (ICT) demonstrating a borderline prolongation and LV ejection time (ET) was significantly shortened in patients with AMI-I compared with normal patients. Perindopril administration induced a borderline shortening of ICT and significant prolongation of ET.*

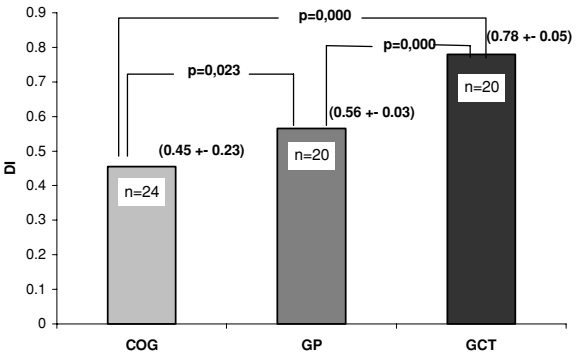

Figure 3. *Comparison of Doppler index (DI) of global myocardial performance between normal patients (COG), patients who received conventional treatment (GCT) for acute inferior myocardial infarction (AMI-I), and patients who received perindopril in addition to conventional treatment (GP). DI was significantly higher in patients with AMI-I compared with normal patients and was improved by perindopril administration.*

the value in GP patients (P = 0.000; Fig. 3, Table II).

Type of LV Diastolic Dysfunction Detected in Patients of Groups Studied

Twelve (30%) of 40 patients with AMI-I preserved normal diastolic performance (8/20 in GP and 4/20 in GCT). Sixteen patients (40%) demonstrated only decreased peak filling rate pattern (8/20 in GP and 8/20 in GCT). Eleven patients (27.5%) had an impaired LV relaxation filling pattern (E/A < 1,[58] DT > 220 msec,[58] IRT > 105msec[58]), 4/20 in GP, and 7/20 in GCT. Only one patient (2.5%) in GCT demonstrated a restrictive LV filling pattern (shortened DT and higher E to A ratio).

Discussion

AMI is characterized by varying degree of LV systolic and diastolic dysfunction. The Tei index, which combines systolic and diastolic time intervals, could be useful for the

Table II

Comparison of Systolic and Diastolic Doppler Parameters as well as Tei Index of 24 Healthy Subjects (GOG), 20 Patients with AMI-Inferior (AMI-I) Receiving Perindopril Plus Conventional Treatment (GP) and 20 Patients with AMI-I Receiving Only Conventional Treatment (GCT).

Doppler Variables	COG (MV ± SE) n = 24	GP (MV ± SE) n = 20	P	COG (MV ± SE) n = 24	GCT (MV ± SE) n = 20	P	GP (MV ± SE) n = 20	GCT (MV ± SE) n = 20	P
ICT (ms)	54.58 ± 2.17	56.84 ± 2.68	NS	54.58 ± 2.17	63.5 ± 3.31	0.021	56.84 ± 2.68	63.50 ± 3.31	NS
ET (ms)	306.46 ± 5.67	274.25 ± 7.35	0.001	306.46 ± 5.67	253.50 ± 7.68	0.000	274.25 ± 7.35	253.50 ± 7.68	0.042
Wave E (m/sec)	0.79 ± 0.03	0.66 ± 0.04	0.011	0.79 ± 0.03	0.60 ± 0.04	0.000	0.66 ± 0.04	0.60 ± 0.04	NS
Wave A (m/sec)	0.68 ± 0.04	0.74 ± 0.05	NS	0.68 ± 0.04	0.74 ± 0.05	NS	0.74 ± 0.05	0.74 ± 0.05	NS
Ratio E/A	1.20 ± 0.04	0.94 ± 0.08	0.020	1.20 ± 0.04	0.91 ± 0.10	0.009	0.94 ± 0.08	0.91 ± 0.10	NS
IRT (ms)	96.04 ± 2.41	120.00 ± 4.23	0.000	96.04 ± 2.41	139.00 ± 6.74	0.000	120.00 ± 4.23	139.00 ± 6.74	0.006
Deceleration time (ms)	195.00 ± 6.68	222.2 ± 11.65	NS	195.00 ± 6.68	227.50 ± 13.91	0.034	222.25 ± 11.65	227.50 ± 13.91	NS
Doppler-index	0.45 ± 0.23	0.56 ± 0.03	0.023	0.45 ± 0.23	0.78 ± 0.05	0.000	0.56 ± 0.03	0.78 ± 0.05	0.000

ICT = isovolumetric contraction time, ET = ejection time, IRT = isovolumetric relaxation time, AMI and statistics as in Table I.

evaluation of the effect of several drugs on the global LV performance in patients with AMI. As was already mentioned in the introduction, the influence of ACE inhibitors on MI has been studied extensively,[1–23] but their favorable effect on small MI is still controversial.[13,14] In this study, we compared the general echocardiographic and Doppler variables as well as the index value of 20 patients with AMI-I treated only with conventional treatment, 20 patients with AMI-I who received perindopril in addition to this therapy, and 24 age- and gender-matched healthy patients.

The isovolumetric relaxation time interval was significantly increased in our patients with MI compared with that in normal patients. This finding is attributable to the development of compensatory hypertrophy during the LV remodeling process after AMI. On the other hand, Poulsen et al.[48] reported no difference between the two groups concerning this time interval. This discrepancy could be explained by the following observations: (1) the Poulsen et al. material also included patients with a history of old MI and more severe ventricular dysfunction, who developed higher LV filling pressures and presented shorter isovolumetric relaxation time compared to our patients; and (2) echocardiographic examination in the Poulsen study was performed during the hyperacute phase of MI (within 1 hour of the patient's arrival at the coronary care unit). It is well accepted that during this period LV systolic dysfunction appears to predominate.[51–53] In our study, patients were assessed in the acute phase (on eighth postinfarct day) when impairment of relaxation had developed as a result of the remodeling process.[51–53] The significant increase of isovolumetric relaxation time was the main cause of prolonged relaxation in most of our patients, supporting our hypothesis that relaxation impairment would be present during the eighth postinfarct day. Perindopril administration shortened IRT, improving LV diastolic function in GP patients compared with GCT patients. It is accepted that ACE inhibitors improve LV diastolic performance[59,60] directly (accelerating myocardial relaxation) and indirectly (lowering systemic arterial pressure). In our study there was no difference between GP and GCT patients concerning arterial blood pressure. Therefore, the favorable impact of perindopril (given in such a low titration) seems to be a direct tissue effect regardless of its hemodynamic action. It is certain that in such a population of patients with preserved systolic LV function, ACE inhibition is not important in the regulation of water and sodium metabolism or in the control of loading conditions (through circulating Renin-Angiotensin-Aldosterone system). Instead, interaction with tissue Renin-Angiotensin and Kinin/Kininases systems seems more relevant.

Regarding the variations in systolic Doppler parameters in our patients, isovolumetric contraction time was prolonged, but not significantly, and ejection time was significantly shortened compared with healthy patients. These findings are consistent with previous studies evaluating time intervals in MI[61,62] and with the Poulsen et al. study.[48] The insignificant increase of isovolumetric contraction time detected in our patients can be explained by the limited systolic dysfunction caused by AMI-I. However, in our previous studies,[46,47] the patients with anterior MI had significantly longer isovolumetric contraction time. Moreover, prolongation of relaxation and subsequent decrease in diastolic filling of LV affected its systolic performance and caused the significant shortening of ejection time observed in our patients. These disorders are due to the existence of infarct area, residual ischemia, stunned myocardium, and remodeling process. Perindopril administration was associated with significant prolongation of ejection time and limited shortening of isovolumetric contraction time, findings attributable to amelioration of the remodeling process, acceleration of relaxation, and improvement of systolic function by ACE inhibition.

These alterations of systolic and diastolic time intervals in patients with AMI-I caused proportional changes in the value of the Doppler index. Thus, prolongation of isovolumetric relaxation and contraction time intervals and shortening of ejection time resulted in a statistically significant rise of the index in patients with AMI-I compared with that in normal patients. This is an indication of global LV function impairment, reflected in index value, in patients after their first uncomplicated AMI-I and is consistent with our previous studies[46,47] and with the Poulsen et al. study.[48] The significant shortening of IRT and prolongation of ejection time occasioned by perindopril resulted in significant reduction of the value of Doppler index in GP patients, compared with that in GCT patients. Therefore, short lasting administration of perindopril, beginning the third postinfarct day, has a favorable impact on global LV performance.

Study Limitations

The Tei index values of the three groups studied could have been influenced by postural changes and preload[63] or afterload alternations. Thus, healthy nonhospitalized patients might have different values compared with postinfarct patients, in supine position, after several days of hospitalization, independently of other factors. However Tei index variation attributable to preload changes seems considerable only in healthy individuals and not in patients with previous MI.[63] Because two of the three groups in our study were constituted from subjects with AMI-I during hospitalization, we consider that the reliability of our results about the beneficial effect of perindopril on Tei index value is not reduced. Regarding the afterload changes, in our material there was no significant difference in blood pressure between the two groups of patients, besides which DI seems to facilitate evaluation of ventricular function irrespective of afterload.[64] Certainly, further studies are needed to clarify this issue.

The ejection time of the Doppler IRT value also distinguished the three groups of our study. However, this time interval is a pure diastolic Doppler parameter, which is depended on heart rate,[30] breathing,[31] age,[32] blood pressure, and preload.[33] On the other hand, Tei index reflects the changes of both IRT and ET, provides information for global LV function, and it is characterized by more advantages[34,35,49,50] than limitations. Larger randomized trials using this index are needed to establish this beneficial effect of ACE inhibitors. In our study, patients underwent M-mode, two-dimensional, and Doppler echocardiography only on the 8.07 $\pm$ 1.16 (SD) postinfarct day, but not later during follow-up, when additional information could have been obtained about the efficacy of these drugs. However, our study suggests that this treatment improves global myocardial performance after AMI of limited extension and the Doppler index serves as a powerful tool for defining prognosis and guiding therapy.

Conclusions

It seems reasonable to conclude that: the global LV function in patients with first uncomplicated AMI-I is impaired in the early postinfarct phase; treatment with perindopril early in the course of AMI-I has a favorable impact on global LV performance, which is apparent in a short time period; and perindopril accelerates relaxation, which indirectly improves systolic function. This beneficial effect derives from its direct tissue and antiischemic effect and not from its hemodynamic action. Our study suggests that the Tei index is a reliable and useful parameter for the determination of treatment strategy and the evaluation of the effect of therapy, especially in patients with global LV dysfunction.

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